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SLEEP DEPRIVATION AND EXERCISE TOLERANCE(U) INDIANA  
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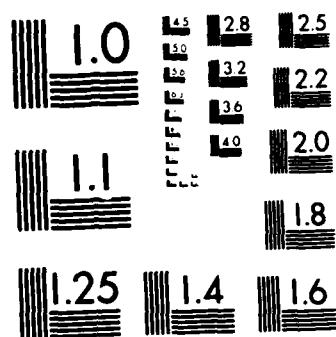
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<p>Acute sleep deprivation radically alters mood and sleepiness. It blunts, as well, the ability to perform long-term maximal exercise. These effects are more evident after 50 than after 25 hours without sleep. Sleeplessness does not, however, change any measured physiological responses to exercise. Stress hormone levels, that is, <math>\beta</math>-endorphin and cortisol, are the same during exercise with or without prior sleep. Similarly, exercise heart rate, ventilation, and blood pressure, and blood lactate levels, are identical under the two conditions. We conclude that sleep loss hampers physical performance primarily through psychological mechanisms.</p>					
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Sleep Deprivation and Exercise Tolerance

Final Report

Bruce J. Martin, Ph.D.

January 1986

Supported by

U. S. ARMY MEDICAL RESEARCH AND DEVELOPMENT COMMAND  
Fort Detrick, Frederick, Maryland 21701-5012

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## SUMMARY

The purpose of the study is to identify the effects of sleep deprivation on the ability of humans to tolerate standard forms of endurance exercise. Standard techniques in human exercise physiology are utilized.

Our results show a) that a 30 hour sleep loss period does not reduce  $\dot{V}O_{2\max}$ , or short-term exercise tolerance, while it leads to a 10% decline in the ability to carry out prolonged exhausting exercise, b) that a 50 hour sleepless period reduces long-term exercise performance about 20%, independent of any clear-cut physiological changes, c) that exercise in severe environmental conditions (cold) is unaffected by a 50 hour sleepless period, d) that sleep loss does not alter the sympatho-adrenal or other stress hormonal responses to subsequent exercise, and e) that sleep loss sizably blunts the ability to produce a maximal or submaximal voluntary ventilation.

We conclude that sleep loss has cumulative effects upon subsequent exercise performance, with these effects being mediated through psychological, not physiological effects.

# FOREWARD

For the protection of human subjects the investigator(s) have adhered to policies of applicable Federal Law 45CFR46.



## BODY OF REPORT

### STATEMENT OF THE PROBLEM

Sleep deprivation is a common occurrence in both the military and civilian spheres. In many instances, severe or prolonged exercise follows sleep loss. Despite this frequent occurrence, little is known of the effects that sleep deprivation may have on subsequent exercise.

### BACKGROUND

Very little was known, prior to initiation of this contract, of either the physiological or psychological effects of sleep loss upon exercise. This deficiency of knowledge extended to both short- and long-term work, and of course nothing was known of the differing effects of various durations and forms of sleep loss. In particular, the influence of sleep deprivation on exercise tolerance was unclear: no previous study had attempted to measure effects in prolonged exercise, and only a few had made measurements in short-term work (1).

Few previous studies had made direct measurements of physiological responses to exercise after sleep. Beyond heart rate, and in some instances, ventilation and oxygen uptake, little was known (2).

Work appearing simultaneous with the active period of this contract has expanded somewhat upon this framework, with particularly exemplary measurements of sweat rate and body temperature rise in the heat after sleep loss (3).

Psychological studies of sleep loss are extensive and detailed, but few have related to the effects of exercise (4). Because some forms and durations of exercise enhance arousal, previous psychological studies are not immediately applicable to the exercise condition.

### APPROACH TO THE PROBLEM

We made a three-pronged attack on the aforementioned dearth of information. First, objective measures of exercise tolerance, both of high-intensity, short-term work and of lower intensity, prolonged exercise, as affected by mild, severe, and fragmentary sleep loss, were made. Second, measurements of the cardiovascular, respiratory, endocrine, thermoregulatory, and metabolic responses to exercise were made. Finally, the psychological responses to the sleep loss exercise combination were assessed to see if exercise alters the psychological influence of sleeplessness, and to evaluate the correlation between performance decrements and psychological alterations.

### RESULTS AND DISCUSSION

Because the results obtained under this contract were grouped into separate studies, they will be discussed in those units and in chronological order:

- 1) Sleep loss for a single night had no effect on maximal or submaximal cycle ergometer exercise oxygen uptake. It also failed to influence sub-maximal exercise  $\dot{V}_E$ , heart rate, or blood pressure. Sleep loss did, for reasons

as yet unclear, reduce slightly but significantly heart rate during short-term high intensity exercise. Performance of that work was unaffected, however, in the six subjects under study. Ratings of perceived exertion were elevated during moderate (50%) and heavy (75% of  $\dot{V}O_{2\max}$ ) exercise after sleep loss, but unaffected during light exercise utilizing 1/4 of maximal oxygen uptake. Measures taken on the day subsequent to the sleep deprivation episode (and after a night of unlimited recovery sleep) showed all parameters at normal levels, including ratings of perceived exertion.

We concluded from this study the following: that short-term performance is unaltered by sleeplessness, that perceptual changes induced by sleeplessness are sizable and enhanced in more demanding work, and that a night of recovery sleep is sufficient to eliminate all measured effects of the deprivation period.

2) Tolerance of prolonged, heavy treadmill exercise was compared with and without a 36 hour sleepless period prior to evening study. Work took place at around 80% of  $\dot{V}O_{2\max}$ , and was reduced after sleeplessness ( $p=0.05$ ) even with doubled monetary incentives for endurance. Individual decrements in performance ranged from less than 5% to 40%; the only apparent correlate of the decline in endurance was elevation of perceived exertion. There was a significantly higher  $\dot{V}_E$  during exercise after sleep loss, though heart rate and metabolic rate were unaffected.

We concluded that prolonged endurance work is hampered by sleeplessness, with no obvious candidate for an explanatory mechanism other than increased perceived exertion at an identical work load.

3) Selection of work load in the normal and sleep deprived conditions was compared in 24 subjects. In these experiments, we asked subjects to adjust their own work rate to provide a "very hard" effort, using treadmill grade as the variable under their control. To our surprise, subjects selected identical work intensities with or without the burden of a previous 24 hour sleepless period, and  $\dot{V}_E$ ,  $\dot{V}O_2$ , and  $\dot{V}CO_2$  were identical as well. There, once again, was a small but significant depression of exercise heart rate after sleeplessness (8 beats/min).

We concluded from this study that ratings of perceived exertion, always elevated after sleep loss, may be a spurious finding, since subjects can be biased by prior expectations of deleterious sleep loss effects. On the other hand, selection of work rate and subjective estimates of fatigue and work intensity are all different, if poorly defined and overlapping, entities, so that these results could not rule out the possibility of perceptual changes in exercise after compromised sleep.

4) Two nights (50 hours) without sleep preceded yet another treadmill walk to exhaustion (65%  $\dot{V}O_{2\max}$ ) and was associated with a 20% diminution of work time. This change was almost, but not quite, significantly greater than that seen after 36 sleepless hours, suggesting a cumulative sleep loss effect on tolerance of long-term heavy exercise. We could find no other evidence of sleeplessness: heart rate, plasma norepinephrine, plasma epinephrine, plasma dopamine, minute ventilation, blood lactate, rectal and mean skin temperature, oxygen uptake and respiratory quotient were all identical with or without sleep loss.

We concluded that the effects of sleep loss on exercise tolerance in long-term work are cumulative, that sleeplessness does not radically alter-- if at all--the sympathoadrenal response to exercise, and that body temperature during work (carried out in a thermoneutral environment) is alike unchanged by loss of sleep.

5) Seven subjects were allowed to exercise to thermal comfort in a very cold ( $0^{\circ}\text{C}$ ,  $2.5 \text{ m}\cdot\text{sec}^{-1}$  air movement) environment, with and without a 50-hour prior sleep loss period. When, despite their exercise, they become too cold to tolerate conditions, the experiment ended. Resting core temperatures were depressed by sleep loss, but returned to control levels within 15-min of exercise. Subjects selected identical work loads for thermal comfort, and became exhausted/miserable after similar period of exposure. Physiological response and thermal perception during the exposure were identical.

We concluded that a profound arousal stimulus, cold as an example, was sufficient to overcome all perceptual and physiological effects of sleep loss, although the duration that these effects might be influential is unclear.

6) Eight subjects worked in either heavy exercise ( $70\% \dot{V}O_{2\text{max}}$ ) or mild exercise ( $25\% \dot{V}O_{2\text{max}}$ ) after 36 hours sleep loss to see if sleeplessness could qualify as a physiological stress at all. In the first phase of this study, sleep was fragmented by numerous random awakenings. Neither protocol elevated or depressed serum cortisol, epinephrine, norepinephrine, or  $\beta$ -endorphin. Subjects' moods were worsened by sleep loss, although prolonged, low-intensity work mitigated this effect.

We concluded from this study that sleep loss does not qualify as a form of physiological stress, though it most certainly is a psychological stress, and if sleep were replaced by exercise rather than waking rest differing results would surely be obtained.

### CONCLUSIONS

Sleep loss has no apparent physiological effects except those on the brain manifested as psychological changes. When exercise is prolonged, demanding, and boring, tolerance will be reduced after sleeplessness.

### RECOMMENDATIONS

Sleep loss is relatively ineffective as a direct inhibitor of exercise tolerance, once the need and motivation for such exercise is established. Its potent effects are psychological, with mood disturbance (toward greater fatigue and confusion) and presumably decreased self-motivation being the primary deficits that influence both the ability to tolerate, and the desire to initiate, exercise.

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